

## Favourable long term prognosis in patients with non-Q wave acute myocardial infarction not associated with specific electrocardiographic changes

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**SUMMARY** Electrocardiograms obtained serially from 544 patients with non-Q wave infarction in the Diltiazem Reinfarction Study were analysed to compare the short term ( $\leq 14$  days) and long term (one year) follow up of 105 patients (19%) whose admission electrocardiogram showed no localisable repolarisation abnormalities (group 1) with the outcome in 439 patients (81%) who had ST-T wave abnormalities (group 2) localised to two or more contiguous leads within an anterior, inferior, or lateral lead group. There were no major between group differences in baseline clinical variables, concomitant medications, or treatment allocation (diltiazem *v* placebo). Group 2 patients, in the first year, had a higher incidence of early recurrent ischaemia (angina  $\geq 24$  hours after myocardial infarction associated with ischaemic repolarisation changes), reinfarction, and readmission for chest pain than group 1 patients, despite comparable creatine kinase and creatine kinase MB activities in both groups.

About 20% of patients with acute non-Q wave myocardial infarction did not have definable ST-T wave abnormalities. These patients had a similar clinical and enzymatic profile as patients with non-Q wave infarction with definable ST-T wave abnormalities and they were more likely to have a favourable short term and long term outcome.

Non-Q wave infarction is defined as acute myocardial infarction without the development of new abnormal Q waves. The electrocardiographic changes associated with non-Q wave infarction are typically ST segment shifts or T wave inversions or both that usually persist for at least 48 hours.<sup>1-3</sup> In earlier studies of non-Q wave infarction, ST-T wave abnormalities without subsequent Q wave evolution were required as a key feature of the diagnosis.<sup>4-15</sup> Many patients with acute myocardial infarction confirmed by creatine kinase MB activity do not show Q waves

or significant repolarisation changes. The frequency of this finding, however, has not been assessed previously.

We recently reported that both the short term<sup>16</sup> and long term<sup>17</sup> outcomes (death and reinfarction) of patients recovering from acute non-Q wave infarction who had angina associated with ischaemic electrocardiographic changes were significantly worse than in those patients without electrocardiographic changes after infarction. Accordingly, we sought to determine whether the frequency, clinical profile, and prognosis of patients who had isoenzyme confirmed non-Q wave infarction *without* definable ischaemic electrocardiographic changes were different from those with electrocardiographic changes.

We used the extensive data base of the prospective

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randomised multicentre Diltiazem Reinfarction Study performed in patients with non-Q wave myocardial infarction.<sup>18,19</sup> The objectives of the present study were: (a) to define the frequency of non-Q wave infarction without localisation of ischaemic electrocardiographic changes on admission, (b) to compare the associated baseline clinical characteristics and enzymatic findings in groups with and without electrocardiographic changes, and (c) to compare the early (2–14 day) and late (1 year) outcome variables (post-infarction angina, reinfarction, death) in patients with and without definable acute electrocardiographic changes on admission.

### Patients and methods

Five hundred and seventy six patients were randomly assigned to receive diltiazem (360 mg/day) or an equivalent placebo within 72 hours of the development of an enzymatically confirmed non-Q wave infarction. The details of the protocol for this multicentre, double blind, randomised trial (Diltiazem Reinfarction Study) have been reported.<sup>18</sup> In brief, the purpose of this trial was to assess whether the prophylactic administration of high dose diltiazem during the early course of non-Q wave myocardial infarction prevented early recurrent infarction (extension) within the first 14 days of hospital admission. Recurrent infarction was defined as a secondary rise in the plasma activity of creatine kinase MB which was analysed every 12 hours throughout hospital stay.

### ELECTROCARDIOGRAPHIC RECORDINGS AND INTERPRETATIONS

The initial diagnosis of acute myocardial infarction was confirmed by serial rises in *both* serum or plasma creatine kinase and creatine kinase MB, according to the laboratory standards for each participating hospital. The electrocardiographic criteria were absence of abnormal Q waves (0.03 second in duration) in  $\geq 2$  leads within a given lead group or absence of R waves ( $\geq 0.04$  second in lead V1 and an R:S  $\geq 1$  in lead V2).

Unlike previous studies, acute ST segment displacement and T wave inversions were not absolute prerequisites for entry into the study. Significant ST segment shifts were defined as the presence of at least 1 mm of ST segment elevation or depression, or T wave inversion in at least two leads within a given infarct location: anterior = leads V1 to V4; lateral = leads I, aVL, V5, and V6; inferior = leads II, III, and aVF. Criteria for posterior non-Q wave infarction could not be established because concomitant anterior ST segment depression in leads V1 to V4 indicative of "anterior"

nontransmural infarction interfered with this electrocardiographic interpretation of early evolving posterior infarction. If there was left ventricular hypertrophy, 2 mm ST segment displacement was needed to establish the site of non-Q wave infarction. For electrocardiographic localisation of a non-Q wave infarction we needed to see changes in at least two leads within one of the three infarct locations (anterior, lateral, or inferior).

The sequential electrocardiograms of all study patients on entry, day 2, day 3, and before discharge were scored and forwarded to the electrocardiographic committee, which consisted of five investigators who were blinded to all clinical information. A total of 2 304 electrocardiograms were analysed and entered into a computerised data bank. All data were stored on the Washington University School of Medicine IBM mainframe computer system and were analysed by the Statistical Analysis System.<sup>20,21</sup>

### CLINICAL AND LABORATORY EVALUATION

After randomisation, the following evaluations were performed: (a) daily clinical evaluation by the investigator; (b) electrocardiogram at the time of recurrent pain and for a minimum of three consecutive days if reinfarction was suspected; and (c) serial blood samples for analysis of total creatine kinase and creatine kinase MB activity. All protocol samples were sent to the creatine kinase core laboratory for measurement of plasma isoenzyme activity by the batch absorption glass bead method.<sup>22,23</sup> An initial sample was taken at the time of randomisation and samples were drawn every 12 hours thereafter throughout the 14 day study.

### DEFINITION OF END POINTS

The primary end point of the Diltiazem Reinfarction Study was reinfarction during the first 14 days after onset of acute non-Q wave infarction. This was defined as an increase of  $\geq 50\%$  above baseline in the plasma activity of creatine kinase MB in at least two samples taken at least six hours apart over 24 hours, with an absolute value  $\geq 15$   $\mu\text{kat/l}$  in at least one sample.

Patients in the Diltiazem Reinfarction Study were followed up by telephone questionnaire of the referring physicians and patient's family. Follow up end points included death, reinfarction, readmission to hospital with recurrent angina, myocardial revascularisation (coronary bypass surgery or percutaneous transluminal coronary angioplasty), or any combination of one or more above cardiovascular events. Follow up was achieved in 535 study participants (91%) and all outcome variables were assessed one year after admission with non-Q wave infarction.

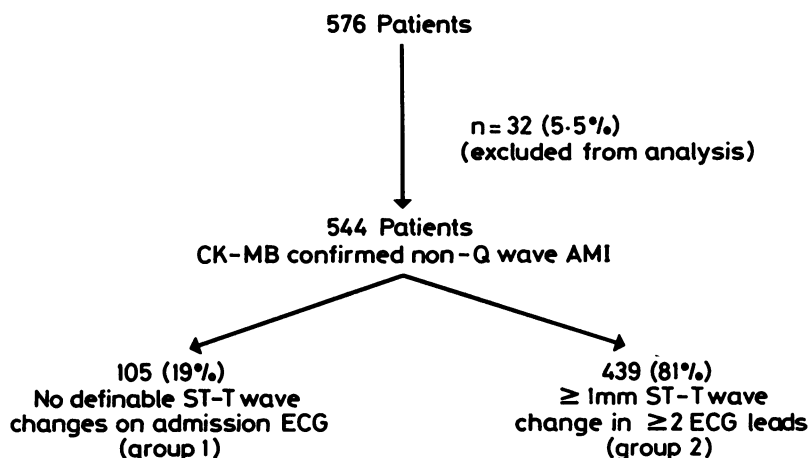


Fig 1 Flow diagram of admission electrocardiographic findings of Diltiazem Reinfarction Study population (see text for description of electrocardiographic criteria). AMI, acute myocardial infarction; ECG, electrocardiogram.

#### STATISTICAL ANALYSIS

Continuous data are expressed as mean (SD). Fourteen day and one year event rates for reinfarction confirmed by creatine kinase MB activity and for death were analysed in the group with ischaemic electrocardiographic changes and the group without. Comparisons between groups were made by *t* tests or the appropriate non-parametric test for continuous variables and Fisher's exact test for discrete variables. A Kaplan-Meier survivorship curve was generated by the LIFETEST procedure of the Statistical Analysis System,<sup>20,21</sup> and one year survival curves were compared by the generalised Wilcoxon test.

## Results

#### CLINICAL CHARACTERISTICS

Thirty two (5.5%) of the 576 patients had evidence of a Q wave myocardial infarction at randomisation (fig 1). These patients were excluded from subsequent analysis. A hundred and five (19.3%) of the remaining 544 study patients with isoenzyme-confirmed non-Q wave infarction had no definable ischaemic changes on the admission electrocardiogram (group 1). The remainder (439 patients (80.7%)) had definable ST-T wave abnormalities on the entry electrocardiogram (group 2). Most of the baseline clinical variables including age, sex, previous myocardial infarction, Killip Class, and blinded treatment assignment were similar in these two groups (table 1). Cigarette smoking was significantly more common in group 2 (57%) than in group 1 (46%).

#### ENZYMATIC FINDINGS

Total mean peak creatine kinase and creatine kinase MB values were assessed for all 544 patients according to the electrocardiographic site of non-Q wave infarction. These subgroup comparisons were based on the location of the repolarisation changes from the qualifying electrocardiogram (table 2). There were no significant differences, although mean total creatine kinase activity was highest in group 1 patients with non-localisable non-Q wave myocardial infarction. Thus even when there were no or non-diagnostic repolarisation changes patients had the same enzymatic findings of myocardial necrosis as those seen in patients with anterior or multiple location non-Q wave infarctions. The mean creatine

Table 1 Baseline characteristics of the two study groups

Characteristic	Group 1 (n = 105)	Group 2 (n = 439)
Age (yr) (mean (SD))	60 (10)	61 (10)
Male (%)	82	77
Obesity (%)	20	21
Cigarette smoking (%)	46	57*
Diabetes mellitus (%)	24	26
Hypertension (%)	42	42
Left ventricular hypertrophy (%)	26	23
Previous AMI (%)	43	42
Previous CABG/PTCA (%)	9	13
Previous CHF (%)	4	7
Killip class		
Class I (%)	79	79
Class II (%)	15	16
Class III (%)	5	6

\*p < 0.05, group 1 v group 2.

AMI, acute myocardial infarction; CABG, coronary artery bypass graft surgery; CHF, congestive heart failure; PTCA, percutaneous transluminal coronary angioplasty.

Table 2 Peak creatine kinase and creatine kinase MB activities (IU/l) according to electrocardiographic location of non-Q wave acute myocardial infarction based on analysis of admission tracing (mean (SD))

Enzyme	Combination ( $\geq 2$ locations)	Lateral (I, aVL, V5-V6)	Anterior (V1-V4)	Inferior (II, III aVF)	Non-localisable (no ST-T change)
CK (IU)	641 (577)	560 (435)	646 (683)	611 (529)	668 (877)
CK MB (IU)	21 (37)	27 (42)	37 (91)	12 (13)	22 (36)

CK, creatine kinase; MB, myocardial band.

p = NS among subgroups by analysis of variance.

kinase and creatine kinase MB values for group 1 patients were 10.7 (14.0)  $\mu$ kat/l (668 (877) IU/l) and 0.22 (0.36)  $\mu$ kat/l (22 (36) IU/l) and for group 2 patients were 10 (9.0)  $\mu$ kat/l (625 (562) IU/l) and 0.24 (0.44)  $\mu$ kat/l (24 (44) IU) respectively (p = NS).

#### SHORT TERM AND ONE YEAR OUTCOME

Twenty one per cent of group 2 patients (93/439) and only 12% of group 1 patients (13/105) had post-infarction angina associated with transient ST-T wave abnormalities before discharge ( $\leq 14$  days) indicative of early recurrent ischaemia (p = 0.040). Reinfarction occurred within 14 days of infarction in 6.6% of group 2 patients (29/439) and in 10.4% of group 1 patients (11/105) (p = 0.172). There were 20 deaths within 14 days of non-Q wave myocardial infarction: 15 in group 2 patients and five in group 1 patients (p = NS).

At one year follow up, 15% of group 2 patients had died (67/439) compared with 8% of group 1 patients (7/105) (p = 0.021) (fig 2). Moreover, 17% of group 2 patients (75/439) developed late recurrent infarction between hospital discharge and one year (17%), compared with 10% of group 1 patients (11/105) (p = 0.043).

Finally, an additional 27% of group 2 patients were readmitted for chest pain during the one year follow up, compared with 20% of group 1 patients (p = 0.056).

#### Discussion

Our results show that approximately 20% of all patients admitted to the coronary care unit with acute non-Q wave myocardial infarction have non-localisable electrocardiographic changes, despite raised

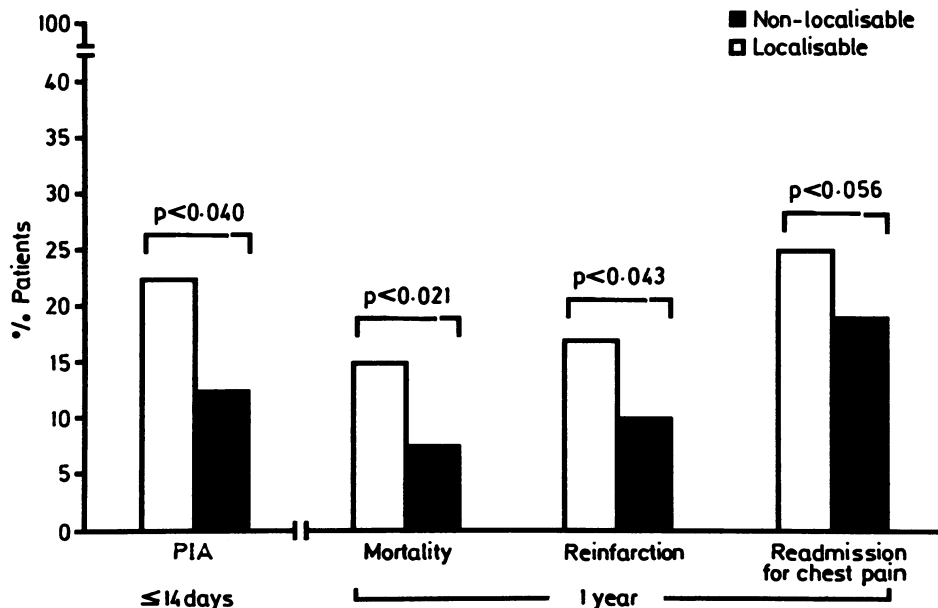


Fig 2 Outcome variables (post-infarction angina (PIA) with transient electrocardiographic changes before hospital discharge, mortality, and reinfarction during one year follow up) for the group of patients with non-Q wave AMI and non-localisable ST-T wave changes and the group of patients with localisable repolarisation abnormalities.

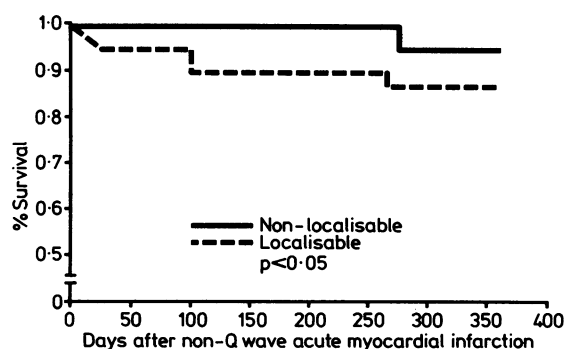


Fig 3 Kaplan-Meier survivorship curve for one year mortality in patients with non-localisable electrocardiographic changes and localisable ST-T wave changes.

activity of plasma total creatine kinase and creatine kinase MB. Early recurrent ischaemia, death within a year of infarction, reinfarction, and readmissions for chest pain were significantly less common in patients without definable ST-T wave abnormalities than in those with.

We found that patients with early post-infarction angina were at increased risk of reinfarction and death during the first two weeks after non-Q wave myocardial infarction,<sup>16</sup> and that angina associated with transient ischaemic electrocardiographic changes identified a subset at particularly high risk. In the subgroup of patients with non-Q wave infarction and electrocardiographic evidence of ischaemia reinfarction was four times more common and the mortality rate within 14 days of randomisation was ten times higher than in patients without early recurrent myocardial ischaemia.<sup>16</sup> We recently extended the period of observation to one year and we confirmed that the presence of post-infarction angina with ischaemic electrocardiographic changes during the hospital stay was a potent predictor of subsequent cardiac events; in these patients mortality and non-fatal reinfarction were significantly increased during the one year follow up.<sup>17</sup>

In the context of our recent observations that ischaemic electrocardiographic changes complicating non-Q wave infarction are a harbinger of adverse short term<sup>16</sup> and long term<sup>17</sup> outcome, the findings in our present report provide important complementary evidence that the *absence* of definable early ST-T wave changes in non-Q wave myocardial infarction characterised a subgroup of patients in whom the risk for late reinfarction and mortality is lower than in those with localisable repolarisation abnormalities. There were no significant differences in peak total creatine kinase or creatine kinase MB

values among subgroups with and without electrocardiographic changes, even in those patients who showed "global" (anterior + inferior + lateral) electrocardiographic changes. Thus the generally more favourable outcome of non-Q wave infarction patients without definable acute electrocardiographic changes could not be explained on the basis of a smaller infarct.

The pathophysiological basis of why almost a fifth of all non-Q wave infarction patients have "electrocardiographically silent" myocardial necrosis was not investigated in the Diltiazem Reinfarction Study. Our findings have several possible explanations. First, when infarction in left ventricular segments is associated with electrocardiographic changes that are electrically opposite in the same plane (anterior-posterior; inferior-lateral) or reciprocal in perpendicular planes (anterior-inferior) the opposing vector forces may cancel out, resulting in a net absence of discernible electrocardiographic findings.<sup>24-26</sup>

Secondly, whereas the process of ventricular depolarisation (phase O) is rapid and attended by a reasonably uniform potential difference across the boundary of activation,<sup>27</sup> repolarisation is considerably longer and non-uniform (phases 2 and 3) and is characterised by differing potentials across various boundaries.<sup>28</sup> Experimentally, the magnitude of T wave changes, unlike that of the QRS complex, has been shown to be unrelated to the mass of myocardium infarcted.<sup>29</sup> Thus patchy areas of necrosis may produce differential regions of repolarisation, resulting in the genesis of non-uniform, absent, or non-specific T waves.<sup>29</sup>

Thirdly, infarction of the posterior wall (or basal lateral segments) of the left ventricle may not be detected with the 12 lead scalar electrocardiogram. This is the last area to be depolarised, and necrosis of this area would affect only the terminal 0.04 to 0.06 second of the QRS complex.<sup>30-32</sup> Such posterior or high lateral infarctions, characteristic of occlusions of the left circumflex or obtuse marginal branch coronary arteries, may be attended by few, if any, electrocardiographic changes.<sup>33 34</sup>

Thus certain patients without definable repolarisation changes could have occult "transmural" infarctions that simply escape electrocardiographic detection. Our observations that this group had higher early peak creatine kinase values, but lower one year mortality and reinfarction rates, support the hypothesis that such patients had "completed" infarctions with less (but certainly not absent) residual myocardial ischaemia.

In summary, there appear to be important prognostic differences between patients with non-Q wave infarction who have definable ST-T wave

abnormalities and those who do not; patients without localisable ischaemic repolarisation changes fared better in the short term and long term than patients with non-Q wave infarction and demonstrable electrocardiographic abnormalities on admission. Nevertheless, rates of early recurrent ischaemia (12%), early reinfarction (10%), and mortality in the first year (8%) were by no means negligible in those without electrocardiographic changes.

Risk stratification techniques can identify subgroups of patients at high risk after infarction who may benefit from additional diagnostic evaluation and more vigorous management.<sup>35 36</sup> Our observations suggest that approximately 20% of patients with non-Q wave myocardial infarction without definable ST-T wave abnormalities are at lower risk and, in the absence of recurrent ischaemic symptoms, may not need the same type of urgent diagnostic/therapeutic intervention before hospital discharge as most other subgroups of patients with non-Q wave infarction. Accordingly, we feel it is appropriate to recommend continuing medical treatment in such patients who are otherwise clinically stable, although regular, careful examinations after infarction would be prudent.

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